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Coupled adaptations affecting cleavage of the VP1/2A junction by 3C protease in foot-and-mouth disease virus infected cells

Maria Gullberg, Charlotta Polacek and Graham J. Belsham

The foot-and-mouth disease virus (FMDV) capsid protein precursor P1-2A is cleaved by the 3C protease to produce VP0, VP3, VP1 and 2A. It was shown previously that modification of a single amino acid residue (K210) within the VP1 protein, close to the VP1/2A cleavage site, inhibited cleavage of this junction and resulted in the production of “self-tagged” virus particles containing the 2A peptide. A second site substitution (E83K) within VP1 was also observed within the rescued virus (Gullberg et al., 2013). It is now shown that introduction of this E83K change alone into a serotype O virus resulted in the rapid accumulation of a second site substitution within the 2A sequence (L2P) that also blocked VP1/2A cleavage suggesting a linkage between the E83K change in VP1 and cleavage of the VP1/2A junction. In a serotype A background, the K210E substitution in VP1 rapidly reverted to wild type. However, introduction of the 2A L2P substitution alone, or with the VP1 K210E change, into this virus resulted in the production of viable viruses. Cells infected with viruses containing the VP1 K210E and/or the 2A L2P substitutions contained the uncleaved VP1-2A protein; the 2A L2P substitution rendered the VP1/2A junction totally resistant to cleavage by 3C protease. The basis for the linkage between amino acid substitutions that are well separated on the surface of the virus particle will be discussed.

Gullberg, M, Polacek, C, Bøtner, A, Belsham, GJ. (2013) Processing of the VP1/2A junction is not necessary for production of foot-and-mouth disease virus empty capsids and infectious viruses: characterization of "self-tagged" particles. J. Virol. 87, 11591-11603.